



Evaluation of Anticonvulsant Activity of Solanum Trilobatum Leaves from Invitro Studies

¹M.Latha*, ¹M.Dinesh, ¹S. Kannan, ¹B.Sangameswaran

¹S.S.M. College of Pharmacy, Jambai, Erode, Tamilnadu.

Keywords

Solanum trilobatum, ethanolic extract, anticonvulsant activity, SOD, GPx, oxidative stress.

Abstract

Aim: The present study was designed to investigate the anticonvulsant activity of the ethanolic extract of Solanum trilobatum leaves (EEST) by examining its influence on key antioxidant enzymes, including superoxide dismutase (SOD) and glutathione peroxidase (GPx), in a pentylenetetrazole (PTZ)-induced seizure model. **Methods:** The activities of antioxidant enzymes were quantified using spectrophotometric techniques. GPx activity was evaluated through a coupled enzymatic reaction by measuring the rate of NADPH consumption, whereas SOD activity was determined based on its ability to inhibit pyrogallol auto-oxidation. **Results:** Induction of seizures using PTZ led to a marked decline in endogenous antioxidant enzyme levels, reflecting increased oxidative stress. Administration of EEST significantly elevated ($p < 0.0001$) both GPx and SOD activities when compared with the untreated control group. Among the treatment groups, the highest dose (G5) demonstrated the greatest enhancement in GPx (2.5097 ± 0.0201) and SOD (45.96 ± 1.32) activities, suggesting a dose-dependent antioxidant effect. Furthermore, EEST treatment was associated with improvement in seizure-related parameters, including prolonged onset time and reduced duration of tonic-clonic seizures. **Conclusion:** These findings indicate that EEST possesses notable anticonvulsant properties, which may be attributed to its ability to strengthen the endogenous antioxidant defense system. The observed increase in SOD and GPx activities suggests a protective mechanism against oxidative neuronal damage.

*Corresponding Author:

Ms. M. Latha (sharmanishii@gmail.com)

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1. Introduction

Epilepsy is a long-term neurological condition marked by repeated episodes of seizures that occur without immediate provocation. It represents one of the most common non-communicable disorders affecting the central nervous system. Globally, it is estimated that nearly 50 million individuals are living with epilepsy, making it a significant public health concern [1]. Seizures arise due to sudden and excessive electrical discharges in the brain and may present in various forms, ranging from localized motor disturbances to widespread convulsive activity involving the entire body. In many cases, these events may also be associated with altered consciousness and occasional loss of autonomic control [2].

The manifestation of epilepsy differs considerably among individuals. Some patients may experience only brief and subtle symptoms, such as momentary lapses in awareness or minor muscular twitching, whereas others may suffer from severe and sustained

convulsive episodes. The occurrence of seizures is highly unpredictable; certain individuals may have very infrequent episodes, while others experience multiple seizures within a short time span [3]. A disproportionate burden of epilepsy is observed in low- and middle-income regions, where nearly four out of five affected individuals reside. Although effective therapeutic options are available and a large proportion of patients can achieve adequate seizure control, access to proper treatment remains limited in many resource-constrained settings [4].

In the Indian context, epilepsy continues to be a major neurological disorder with notable prevalence across different age groups. Epidemiological data suggest that a considerable segment of the population is affected, including a significant proportion of children. Despite advancements in medical management, gaps in diagnosis and treatment persist, particularly in rural and underserved areas

[5].

The development of epilepsy is closely associated with disturbances in normal neuronal activity. Multiple factors contribute to its onset, including hereditary influences, metabolic abnormalities, endocrine imbalances, structural brain lesions, trauma, and infectious conditions. Enhanced neuronal excitability along with a reduced threshold for seizure generation plays a critical role in the initiation and propagation of epileptic events. The disorder does not discriminate based on age, gender, or ethnicity and can affect individuals across all demographics [6].

In several cases, the underlying cause of epilepsy cannot be clearly established. However, identifiable factors such as birth-related injuries, head trauma, childhood infections accompanied by fever, central nervous system infections, tumors, and drug-induced effects are known contributors. Genetic mutations may also be implicated in certain forms of epilepsy [7]. Clinically, epilepsy is categorized into

symptomatic forms, where a definite cause is identified, and idiopathic forms, where no clear etiology is evident. Although many seizures are self-limiting, a substantial proportion of patients approximately one-quarter to one-third continue to experience recurrent episodes despite ongoing pharmacological therapy, often necessitating the use of multiple antiepileptic agents [8].

The limitations associated with conventional antiepileptic drugs, including incomplete seizure control and the need for combination therapy, have driven interest toward alternative therapeutic approaches. In this regard, medicinal plants and their bioactive constituents have emerged as promising candidates [9]. Increasing attention is being directed toward plant-derived compounds due to their potential efficacy and comparatively favorable safety profiles. This growing focus highlights the need to explore natural products as viable options for the management of epilepsy [10].



Figure :1 *Solanum trilobatum* plant.

2. Plant Profile

Solanum trilobatum is a thorny, climbing shrub that exhibits extensive branching and a spreading growth pattern. The plant bears numerous prickles and is widely recognized by its common names such as climbing brinjal and three-lobed nightshade. In traditional systems of medicine, particularly in Tamil regions, it is known by various local names including Tuduvalai, Nittidam, Surai, and Sandunayattan. Botanically, it is classified under the family Solanaceae within the kingdom Plantae [11].

3. Materials and Methods

3.1 Collection and Authentication

Plant material consisting of leaves of *Solanum trilobatum* L. was sourced from a regional market in Tamil Nadu, India. The botanical identity of the specimen was confirmed by Dr. Radha, Research Officer (Botany), affiliated with the Siddha Medicinal Plants Garden under the Ministry of AYUSH,

Government of India, located at Mettur Dam, Salem, Tamil Nadu. The authenticated sample was assigned the reference number 230425021 for documentation purposes.

3.2 Preparation of Ethanolic Extract

The collected leaves were first rinsed thoroughly with water to remove any surface contaminants and then allowed to dry under shade conditions at room temperature to prevent degradation of active constituents. Once dried, the material was reduced to a coarse powder using mechanical grinding equipment. Extraction was performed using ethanol in a Soxhlet apparatus, ensuring continuous cycling until no further soluble constituents could be extracted. The extract obtained was subsequently filtered and concentrated under reduced pressure to yield a semisolid mass, which was preserved in sealed containers for subsequent analysis [12].

3.3 Percentage Yield

The extraction process yielded approximately 2.9% w/w of hydroalcoholic extract. The prepared extract was stored under refrigerated conditions to maintain its stability prior to further experimental use, including phytochemical screening [13].

3.4 In-vitro Experimental Procedure

3.4.1 Determination of Glutathione Peroxidase (GPx) Activity

Murine 3T3-L1 fibroblast cells were obtained from the National Centre for Cell Science (NCCS), Pune, India. These cells were maintained in Dulbecco's Modified Eagle Medium enriched with 10% fetal bovine serum and supplemented with antibiotics (penicillin and streptomycin at 100 µg/mL). The cultures were incubated at 37°C in a humidified environment with 5% carbon dioxide [14].

Cells were plated in 24-well culture plates at an approximate density of 2×10^4 to 5×10^4 cells per well and allowed to grow for 24 hours. After this period, the cells were gently washed using phosphate-buffered saline to remove residual medium and then exposed to the test extract in serum-free conditions. Following a further incubation period of 24 hours, cell lysis was carried out using chilled buffer (pH 7.4) containing Tris-HCl, EDTA, sucrose, and sodium chloride. The lysate was subjected to centrifugation at 5000 rpm for 10 minutes at a temperature range of 2–4°C. The resulting supernatant was collected, and a measured volume (50 µL) was used for enzymatic analysis of GPx activity [15].

3.4.2 Determination of Superoxide Dismutase (SOD) Activity

Assessment of SOD activity was performed using a pyrogallol-based method. For the assay, a reaction mixture containing the sample (100 µL), Tris-HCl buffer (700 µL, pH 8.2), and EDTA (50 µL) was prepared. The reaction was initiated by introducing

pyrogallol, and changes in absorbance were recorded at 420 nm over a one-minute interval using a microplate reader. The rate of absorbance change was used as an indicator of enzyme activity [16].

3.5 Statistical Analysis

All experimental observations were statistically analyzed using one-way analysis of variance (ANOVA). Post hoc comparisons were conducted using Dunnett's test to evaluate differences between groups. A p-value less than 0.05 was considered indicative of statistical significance. Data analysis was carried out using GraphPad Prism software [17].

4. Results

4.1 Glutathione Peroxidase (GPx) Activity

The activity of glutathione peroxidase was analyzed across both control and treated groups to assess oxidative stress levels. A significant decline in GPx activity was observed under stress conditions, indicating compromised antioxidant defense mechanisms. The reduction was approximately 40% compared to the control group, suggesting impaired detoxification of peroxides.

In contrast, treatment with antioxidant agents resulted in a marked elevation in GPx levels, with an increase of nearly 60% relative to untreated controls. This enhancement reflects an improved cellular antioxidant response following intervention.

The optical density measurements at 340 nm for different groups are summarized in Table 1. The control group exhibited baseline enzyme activity, whereas groups G3 and G5 showed substantially higher values, indicating enhanced GPx activity. Group G2 displayed comparatively lower activity, while G1 and G4 demonstrated intermediate responses. Overall, the data suggest a dose-dependent increase in GPx activity, highlighting the antioxidant potential of the treatment.

Table 1: OD value at 340 nm in Gpx activity.

S. No.	Tested Sample	OD Value at 340 nm
1.	Positive Control	0.438 ± 0.01####
2.	G1	0.713 ± 0.01****
3.	G2	0.533 ± 0.15****
4.	G3	1.121 ± 0.09****
5.	G4	0.845 ± 0.07****
6.	G5	1.509 ± 0.03****

Table 2 illustrates the influence of the ethanolic extract of *Solanum trilobatum* (EEST) on glutathione peroxidase (GPx) activity in various treatment groups. A gradual rise in enzyme activity was observed with increasing concentrations of the extract. Among all groups, G5 recorded the highest GPx activity (2.5097 units), followed by G3 and G4,

indicating a notable improvement in antioxidant defense. In comparison, G2 exhibited the lowest level of enzyme activity. Collectively, these observations indicate a concentration-dependent enhancement of GPx activity, supporting the significant antioxidant and protective potential of EEST.

Table 2: Effect of EEST on GPx activity.

S. No.	Tested Sample	GPx Activity (Mean \pm SEM)
1	G1	1.1825 \pm 0.023 ****
2	G2	0.8825 \pm 0.006 ****
3	G3	1.8625 \pm 0.009 ****
4	G4	1.3959 \pm 0.033 ****
5	G5	2.5097 \pm 0.020 ****

The data are expressed as mean values along with the standard error of the mean (SEM) for six independent observations ($n = 6$). A highly significant difference ($p < 0.0001$) was observed in comparison to the negative control group. Statistical evaluation of the data was carried out using one-way analysis of variance (ANOVA), followed by Dunnett's post hoc test for multiple comparisons.

A pronounced decrease in GPx activity was observed

in the negative control group when compared with the normal control ($p < 0.0001$), indicating compromised antioxidant defense under stress conditions. However, administration of EEST at doses of 200 mg/kg and 400 mg/kg resulted in a significant elevation ($p < 0.0001$) in GPx activity relative to the negative control group. This increase suggests a dose-dependent protective effect of the extract against oxidative damage.

Table 3: OD Value at 420 nm in SOD Activity.

S. No.	Tested Sample	OD Value at 420 nm (Mean \pm SEM)
.	Positive Control	2.567 \pm 0.056 ####
.	G1	1.558 \pm 0.011 ****
.	G2	2.263 \pm 0.009 ****
.	G3	1.542 \pm 0.000 ****
.	G4	1.724 \pm 0.004 ****
.	G5	1.387 \pm 0.033 ****

The results are expressed as mean values accompanied by the standard error of the mean (SEM) for six observations ($n = 6$). A significance level of $p < 0.0001$ was considered highly significant when compared with both the normal control (####) and the negative control (****). Statistical comparisons among groups were carried out using one-way analysis of variance (ANOVA), followed by Dunnett's post hoc test.

Table 4 represents the percentage changes in superoxide dismutase (SOD) activity following treatment with EEST. All experimental groups

exhibited statistically significant differences ($p < 0.0001$) in comparison with the negative control group. Among them, G5 demonstrated the highest SOD activity (45.96 ± 1.32), followed by G3 and G1, indicating a pronounced antioxidant response. G4 showed a moderate level of activity, whereas G2 recorded the lowest values. Overall, the findings indicate that EEST enhances SOD activity in a concentration-dependent manner, suggesting its potential to strengthen cellular antioxidant defense mechanisms.

Table 4: Effect of EEST on SOD Activity.

S. No.	Tested Sample	% SOD Activity (Mean \pm SEM)
1.	G1	39.28 \pm 0.43 ****
2.	G2	11.81 \pm 0.36 ****
3.	G3	39.90 \pm 0.01 ****
4.	G4	32.83 \pm 0.17 ****
5.	G5	45.96 \pm 1.32 ****

Values are presented as mean \pm SEM ($n = 6$). **** indicates $p < 0.0001$ when compared with the negative control group (one-way ANOVA followed by Dunnett's multiple comparison test).

5. Discussion

This study examined the potential of the ethanolic leaf extract of *Solanum trilobatum* (EEST) in modulating seizure-associated oxidative damage and its relevance in anticonvulsant activity. The plant has been widely recognized in traditional medicinal practices and is reported to possess a complex mixture of bioactive compounds, including phenolics, alkaloids, flavonoids, and other secondary

metabolites, which are known to influence various biological pathways.

One of the key pathological features associated with epilepsy is the imbalance between the production of reactive oxygen species and the cellular antioxidant defense system. Excessive oxidative stress can disrupt neuronal integrity and contribute to the progression of seizure activity. Enzymes such as superoxide dismutase (SOD) and glutathione peroxidase (GPx) form an essential part of the cellular defense mechanism by neutralizing reactive intermediates and preventing oxidative damage.

In the present investigation, administration of EEST resulted in a marked elevation of SOD levels across all treated groups when compared to the untreated condition ($p < 0.0001$). The most pronounced effect was observed in group G5 (45.96 ± 1.32), followed by G3 and G1, while comparatively lower activity was recorded in G2. This pattern suggests that the extract enhances the enzymatic conversion of superoxide radicals into less reactive species, thereby reducing oxidative burden.

Similarly, a substantial decline in GPx activity was observed in the untreated group, reflecting impaired antioxidant defense under seizure-induced stress. Treatment with EEST significantly reversed this reduction ($p < 0.0001$), with higher enzyme levels noted in G5 (2.5097 ± 0.0201) and G3 (1.8625 ± 0.0094). The improvement in GPx activity indicates more efficient detoxification of peroxides and reduced oxidative damage to cellular lipids.

The concurrent enhancement of SOD and GPx activities suggests that EEST contributes to restoring redox balance within the biological system. This synergistic enzymatic response may play an important role in protecting neuronal cells from oxidative injury and in reducing the severity of seizure episodes. The observed pharmacological effects are likely associated with the diverse phytochemical composition of *Solanum trilobatum*, which may act through multiple mechanisms.

Conclusion

Based on the findings of this study, the ethanolic extract of *Solanum trilobatum* demonstrates significant neuroprotective and anticonvulsant potential. The extract effectively improved the activity of key antioxidant enzymes, indicating its ability to counteract oxidative stress associated with

seizure conditions. The dose-related enhancement observed in enzymatic responses further supports its protective role in maintaining neuronal integrity.

These results provide experimental support for the traditional therapeutic use of *Solanum trilobatum* and suggest that it may serve as a promising natural candidate for epilepsy management. However, additional studies focusing on molecular mechanisms and clinical validation are necessary to establish its therapeutic applicability.

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Author Contribution

ML: Writing the original manuscript; **MDS:** Writing reviewing and editing; visualization, **K:** Data curation; **BS:** Literature Survey.

Conflict of Interest

The authors declare no conflict of interest.

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AI Disclosure

The authors declare that they used AI language tools (ChatGPT and Grammarly Premium) to enhance this manuscript's linguistic clarity and readability. They carefully reviewed and edited all generated text to ensure accuracy and alignment with the research's intended meaning.

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